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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Application Number: 10/677,733
Filing Date: October 01, 2003
Appellant(s): GARDNER ET AL.

Richard Aaron Osmond
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed 25 November 2008 appealing from the Office action mailed 06 August 2008.

(1) Real Party of Interest

A statement identifying by name the real party of interest is contained in the brief.

(2) Related Appeals and Interferences

The following are the related appeals, interferences, and judicial proceedings known to the examiner which may be related to, directly affect or be directly affected by or have a bearing on the Board's decision in the pending appeal:

US Application No: 10677733 (which is also the instant case on appeal) - Instant claims 3 and 5 correspond to canceled claim 1 (6/21/06), and instant claim 4 corresponds to canceled claim 2 (6/21/06). Claim 1 has been before the Board of Patent Appeals and Interference (BPAI) - said claim was rejected under 35 U.S.C. 103(a) using the exact same art and the nearly the exact same reasoning as presented thus far and herein. The BPAI Affirmed the examiner's rejection under 103(a), however, said BPAI inserted additional reasoning of their own. It was stated (see Board Decision of 19 September 2007, p. 5, 3rd paragraph):

> "In reaching this conclusion, we acknowledge that the Examiner erred in finding that "the PAS domains of the cited [prior] art must contain a binding cavity" (Answer 6; see Reply Br. 3, stating that the Examiner's assertion is "contrary" to the evidence of record). However, we do not find this misstatement fatal to the rejection. Nonetheless, because we have supplemented the rejection with reasoning of our own, we designate it as a new ground of rejection under 37 C.F.R. § 41.50(b)."

(3) Status of Claims

The statement of the status of claims contained in the brief is correct.

(4) Status of Amendments After Final

No amendment after final has been filed.

(5) Summary of Claimed Subject Matter

The summary of claimed subject matter contained in the brief is correct.

(6) Grounds of Rejection to be Reviewed on Appeal

The appellant's statement of the grounds of rejection to be reviewed on appeal is correct.

(7) Claims Appendix

The copy of the appealed claims contained in the Appendix to the brief is correct.

(8) Evidence Relied Upon

5,843,683	EDERY et al.	12-1998
6,436,654	BERKENSTAM et al.	08-2002
6,291,429	TAKAHASKI et al.	09-2001
WO 97/18471	FESIK et al.	05-1997

Board of Patent Appeals and Interference Decision in Appeal 2007-2956, which is from the instant application (10677733) - decided 19 September 2007.

Taylor et al. (Microbiology and Molecular Biology Reviews, 1999, 63(2):479-506).

In re Farrenkopf , 713 F.2d 714, 219 USPQ 1 (Fed. Cir. 1983).

(9) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

Claims 3 and 5 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Fesik (WO 97/18471) in view of any one of U. S. patents 5,843,683 (Edery *et al.*); 6,291,429 (Takahaski *et al.*); or 6,436,654 (Berkenstam *et al.*) for the reasons of record which are reiterated below - (also see previous Office actions from: 11 December 2007, 05 September 2006 and 21 February 2006 and the Board of Patent Appeals and Interferences decision of 19 September 2007, which affirms the Final rejection of 05 September 2006 for claim 1 (which corresponds to instant claims 3 and 5)).

Fesik teaches a method of identifying compounds that bind proteins using NMR methods, which include comparing the NMR of $^{1}H/^{15}N$ correlation spectra of ^{15}N labeled protein in the presence and absence of a potential compound that binds to said protein, see abstract and page 7, lines 29-32. They motivate one of ordinary skill in the art to use their method as they teach the many advantages of using their method, see page 8,

line 8 to the end of the page. Also, they teach the application of their method to several proteins, see examples 1-2 and 4. Thus, the method is applicable to any protein of interest. Fesik does not teach the application of his method to identify compound that bind or interact with PAS domain or proteins.

Edery *et al.* teach that abnormalities in PAS domain protein function may cause certain conditions or diseases in human, such as human behavior disorders and epithelial tissue cancer, see column 1, lines 41-55. Also, they teach that xenobiotics such the aryl hydrocarbon or dioxin complex (AH) with receptor containing PAS domain activates the metabolism of the xenobiotics in the liver and lungs of mammals, but the activation process produces gene products which are able to convert the xenobiotics to carcinogens, see column 2, lines 22-40. Thus, it appears that compounds that modulate the activity of the PAS domain would be useful in the prevention and treatment of disease, see column 3, lines 47-56.

Takahaski *et al.* teach the human and mouse genes and polypeptides component of the circadian clock (the clock polypeptide), which are member of the basic helix-loop-helix-PAS domain family of proteins, see column 7, lines 53-65. The polypeptide is thought to be involved in many regulatory functions in humans, see column 16, line 8 through column 21, line 27. Also, Takahaski *et al.* teach that modulator of the clock polypeptide can be used for the identification of drugs for the treatment of circadian rhythm dysfunctions, see column 9, lines 13-27.

Berkenstam *et al.* teach the various domains encompassed in human HIF-1α including PAS-B residues, 178-390, of the human protein and its function, see column 7, last paragraph and column 8, lines 31-37 and column 11, lines 11-62. Also, they teach that compounds that modulate the activity of various domains are potentially useful in the regulation of target genes normally associated with HIF-1α such as genes involved in angiogenesis, erythropoiesis, and glycolysis.

Each of Edery *et al.,* Takahaski *et al.* and Berkenstam *et al.* provide one of ordinary skill in the art with motivation to identify modulator of the PAS domains in various protein as they teach modulator of the PAS domain are potential drugs to prevent and treat serious diseases. Fesik provide one of ordinary skill in the art with motivation to use their method to identify ligands for the PAS domains as they teach an easy method amenable to automation for identification of modulator of protein activity. Thus, it would have been obvious to one of ordinary skill in the art to use the method taught by Fesik to identify potential compound that modulate the activity of the PAS domain protein. Thus, the claimed invention was within the ordinary skill in the art to make and use at the time it was made and was as a whole, *prima facie* obvious.

(10) Response to Argument

Appellants' augments and the declarations presented by Professor's Stephen Sprang and Kevein H. Gardner, both filed under 37 C.F.R. 1.132 have been fully considered, but they are found to be unpersuasive.

The scope of the rejected claims 3 and 5 remain consistent with the scope of previous claim 1 (now canceled) which was rejected under 35 U.S.C. 103(a) utilizing the exact same references presented for instant claims 3 and 5. The Board of Patent Appeals and Interferences (BPAI) affirmed the Examiner's rejection of claim 1 as being unpatentable over Fesik (WO 97/18471) in view of any one of U. S. patents 5,843,683 (Edery *et al.*); 6,291,429 (Takahaski *et al.*); or 6,436,654 (Berkenstam *et al.*). The Board stated the following in their decision of 19 September 2007 (see p. 6:

"Appellants contend that:

> the prior work provided no evidence of cofactors for most PAS domains, and taught that those limited PAS domains having cofactors required them for proper

> folding, and taught that PAS domains without cofactors had tightly packed cores with no pre-formed cavities that would suggest a cofactor or ligand binding site, one skilled in the art would not have suspected that such PAS domains (without known cofactors and having tightly packed cores with no pre-formed cavities that would suggest a cofactor or ligand binding site) would be rational candidates to screen for core ligand binding; in fact, the art (supra) teaches squarely away from such use.
> (App. Br. 5.)

We do not agree that "one skilled in the art would not have suspected that... PAS domains (without known cofactors and having tightly packed cores with no pre-formed cavities that would suggest a cofactor or ligand binding site) would be rational candidates to screen for core ligand binding" (Appeal Br. 5). Takahaski suggests a method for identifying ligands for a PAS protein having a hydrophobic core (Answer 6). Edery also describes an assay method for identifying compounds that regulate a PAS domain protein's activity. Thus, despite the fact that these proteins have tightly packed cores with no pre-formed cavities - a fact that Appellants have not challenged - it was still suggested that these PAS domain proteins be utilized for ligand screening (see, e.g., Takahaski, at col. 9, ll. 14-16; Edery, at col. 46-50)."

In making its conclusions regarding the previous decision, the Board also stated the following: (see Board Decision of 19 September 2007, p. 5, 3rd paragraph):

> "In reaching this conclusion, we acknowledge that the Examiner erred in finding that "the PAS domains of the cited [prior] art must contain a binding cavity" (Answer 6; see Reply Br. 3, stating that the Examiner's assertion is "contrary" to the evidence of record). However, we do not find this misstatement fatal to the rejection. Nonetheless, because we have supplemented the rejection with reasoning of our own, we designate it as a new ground of rejection under 37 C.F.R. § 41.50(b)."

Thus, a case of *prima facie* obviousness to combine the teachings of the prior art, specifically, Fesik et al. in view of any U.S. patents 5,843,683 (Edery *et al.*); 6,291,429 (Takahaski *et al.*); or 6,436,654 (Berkenstam *et al.*) has been established by the Board itself.

Appellants thus turn to what they deem as the Board's perceived deficiencies in reasoning in reaching their previous conclusion of affirmation of the 35 U.S.C. 103(a) rejection of record and present these in the arguments of the instant Appeal Brief.

Appellants acknowledge the following regarding which was well known in the art prior to filing the instant application (see instant Appeal Brief, p. 4, 2nd full paragraph):

(i) It was known to use NMR to identify ligand binding to target molecules.

(ii) PAS domains are protein interaction domains widely used for intra- and intermolecular associations.

(iii) There were two structurally and functionally distinct classes of PAS domains:

(a) one kind (e.g. PYP PAS) purifies with a core-bound cofactor required for proper folding and formation, wherein crystallographic analysis showed the cofactor bound inside a core pocket; and

(b) the other class of PAS domains (e.g. HERG PAS) did not purify with a core-bound ligand, wherein crystallographic analysis revealed a tight core with no apparent core binding pocket.

Appellants also acknowledge that it is the latter class (e.g. (b)) which is the subject of the instant claims and further stipulate:

"we whole-heartedly agree with the Board's conclusion that the prior art suggests utilizing this class of PAS domains (without bound cofactors and showing tightly packed cores with no pre-formed cavities) for ligand screening:" (see instant Appeal Brief, p. 4, 2nd full paragraph, last line)

Appellants also acknowledge the prior art taught that the N-terminal PAS domain of the HERG potassium channel was postulated to self-regulate the HERG protein by binding the channel body and state (see instant Appeal Brief, p. 4, last paragraph to p. 5, 1st paragraph):

> "we similarly whole-heartedly concur with the prior Board Decision's conclusion that it would have been obvious to try to target this activity with regulatory small molecules."

It is stated, however, that the dispute that Appellants have is in exactly what kind of screening assays the prior art suggests. Appellants thus summarize the various screening assays which the identified prior art teaches. It is noted that Morais Cabral used scanning mutagenesis to identify a hydrophobic patch on the surface of the HERG PAS domain that forms an interface with the body of the potassium channel to which it tightly binds (see instant Appeal Brief, p. 5, last paragraph to p. 6, 1st paragraph). It is acknowledged that the cited Edery (US 5,843,683) and Takahaski (US 6,291,429) also propose screening compounds in functional assays (see instant Appeal Brief, p. 6, 2nd paragraph). In particular, it is asserted that Edery proposes *in vivo*, transcriptional reporter assays for PAS dimerization (see instant Appeal Brief, p. 6, 3rd paragraph). The cited Berkenstam (US 6,436,654) similarly discloses methods for identifying compounds which modulate the "function" of HIF-la wherein the only specifically disclosed assay is a transcriptional reporter read-out assay (see instant Appeal Brief, p. 6, 4th paragraph). And finally, Takahaski proposes inhibiting functional activity of the CLOCK polypeptide, such as DNA binding (see instant Appeal Brief, p. 6, last paragraph to p. 7, 1st paragraph).

It is summarily argued that no where do any of these references suggest screening for small molecules that simply bind the subject PAS domains. A small molecule that simply bound the subject PAS domain would be of no interest to the authors of the cited art. Hence, Appellants can not agree that these references suggest

screening for compounds in an *in vitro* binding assay format, such as NMR (see instant Appeal Brief, p. 6, last paragraph to p. 7, 2nd paragraph).

However, the references relied upon are merely indicative of the state of the prior art which is many different screening assays are performed on the extremely well known PAS domain proteins. Further, the rationale of screening the well known PAS domains using a technique such as NMR as taught by Fesik et al., is that it is a quick and efficient method of screening small molecules and identifying/detecting in the candidate protein they might bind. As Edery *et al.* teach, it is known that abnormalities in PAS domain protein function may cause certain conditions or diseases in humans, such as human behavior disorders and epithelial tissue cancer (see column 1, lines 41-55). Thus, it appears that compounds that modulate the activity of the PAS domain would be useful in the prevention and treatment of disease (see column 3, lines 47-56). Berkenstam et al. similarly teach that the PAS-B domain of Hif-1α and compounds that modulate its activity are potentially useful in the regulation of various genes involved in angiogenesis, erythropoeisis and glycolysis. Therefore, for Appellants to state that "A small molecule that simply bound the subject PAS domain would be of no interest to the authors of the cited art" is entirely contrary to the teachings of Edery et al. Berkenstam et al. and why one skilled in the art would precisely be motivated to screen for compounds that bind to PAS domains.

Furthermore, if it is known that a protein of interest, in this case one with a PAS domain, does or does not have various *in vitro* or *in vivo* activities in the presence or absence of certain ligands or cofactors which contribute the proteins overall function,

then one skilled in the art would reasonably conclude that it must therefore likely bind the protein/PAS domain somewhere on the protein. Given the fact that many PAS domains DO require cofactors to initiate allosteric control of the proteins function (e.g. FixL which uses the heme to detect oxygen levels, see specification p. 1), one skilled in the art would also reasonably conclude that other co-factors or ligands may be required by other PAS domains, even those without apparent ligand binding sites. In addition, the prior art established that it was accepted that PAS domains are signaling modules that monitor changes in environmental conditions *via core-bound molecules* (see Taylor et al, 1999; pg 480, parg 1; pg 488, parg 3; pg 490, parg 2, 1st column). Thus utilizing various screening methods, especially the method of NMR as taught by Fesik et al. which has significant advantages in performing *in vitro* ligand binding screening, would be obvious given the teachings of Edery, Takahashi or Berkenstam et al. which provide clear motivation to one skilled in the art to screen for ligands that modulate the activity of proteins having PAS domains.

Appellants finally state that for "good measure" they provide further affirmative evidence which documents that one skilled in the art would have considered the claimed invention non-obvious and thus provide the declarations of Professors Stephen R. Sprang and Kevin H. Gardner.

While the Examiner believes that a case of *prima facie* obviousness has already been established, the declarations will be addressed below.

With respect to the affidavit provided by Prof. Sprang, it is noted that the majority of the evidence provided within said declaration is a reiteration of known facts (e.g. PAS

domains are one of the most studied and documented protein domains – Point 2, p. 1; a foreign ligand of a PAS domain is distinct from a natural ligand and that the claims require the core has no a priori NMR apparent cavity - Point 3, p. 1-2), which culminates in the conclusion that since there was no evident a priori ligand binding site one skilled in the art would not have found the claims obvious in light of the sited references.

These assertions and the reasons for finding contrary have been presented by the Board itself in the previous decision set forth on 19 September 2007.

The Declaration under 37 CFR 1.132 by Professor Kevin H. Gardner, filed 11 April 2008, is also believed to be insufficient to overcome the instant rejection of claims 3 and 5 based upon 35 U.S.C. 103(a) because of the following reasons.

Professor Gardner asserts that the previous Office action (11 December 2007) is not reflective of the ordinary skill and understanding in the art and that "Its conclusions are unwarranted and erroneous, and its analysis contains multiple and fundamental overstatements and technical inaccuracies." (see Declaration, point 3). In support of these assertions, Prof. Gardner states that the Office action relies on two suppositions which are both technically incorrect; that being that it would be obvious to screen PAS domains because they all contain ligand binding sites and that solution NMR is an obvious choice for a screening method to identify the protein-binding ligands for any target (see point 4, p. 1). It is asserted that the Examiner's argument that reasons a skilled artisan would have known of the presence of the ligand-binding cavity because the protein has an activity in solution, is misleading and inaccurate (see point 5, pp. 1-2). Prof. Gardner asserts that most PAS domains are not naturally regulated by small

molecule ligands or cofactors and that the art teaches that most PAS domains function as constitutive protein/protein interaction domains in their current settings, independent of small molecule regulation (see point 5, p. 2).

However, it is asserted that this is neither a misleading or inaccurate statement. If it is known that the protein of interest does or does not have various activities in the presence or absence of certain ligands or cofactors which contribute the proteins overall function, then one skilled in the art would reasonably conclude that it must therefore likely bind to the protein/PAS domain (also see previous Office action, p. 3, last paragraph to p. 4). In addition, given the fact that many PAS domains do require cofactors to initiate allosteric control of the proteins function (e.g. FixL which uses the heme to detect oxygen levels, see specification p. 1), one skilled in the art would also reasonably conclude that other co-factors or ligands may be required by other PAS domains. In addition, given what was known in the art at the time of filing, it was accepted that PAS domains are signaling modules that monitor changes in environmental conditions via core-bound molecules (Taylor et al, 1999; pg 480, parg 1; pg 488, parg 3; pg 490, parg 2, 1st column). Thus, Taylor et al. teach that, more likely than not, any PAS domain will bind a ligand in its core. Finally, the position taken by the Board of Patent Appeals and Interference was that they did not agree that one skilled in the art would not have suspected that PAS domains (without known cofactors and having tightly packed cores with no pre-formed cavities that would suggest a cofactor or ligand binding site) would be rational candidates to screen for core ligand binding" because Takahaski suggests a method for identifying ligands for a PAS protein having a

hydrophobic core, Edery describes an assay method for identifying compounds that regulate a PAS domain protein's activity. Thus, they conclude that despite the fact that these proteins have tightly packed cores with no pre-formed cavities it was still suggested that these PAS domain proteins be utilized for ligand screening (see, e.g., Takahaski, at col. 9, ll. 14-16; Edery, at col. 46-50) - See BPAI decision, p. 6.

Prof. Gardner also asserts that while the accepted view in the field is that developing protein/protein inhibitor is difficult but not impossible, the instant invention is nonetheless non-obvious because core targeting of inhibitors raises additional challenges. Namely, a) without an *a priori* formed cavity there would be an overwhelming expectation that our targeted core ligand binding sites would not even exist; b) it would be uncertain and unpredictable whether ligand binding to interior sites could provoke an allosteric change that affects a proteins function; and c) compounds that target internal sites bind much slower and thus possess slower "on" rates.

However, given that proteins in solution are not static and "breathe" while in solution, if one understands that a given protein may bind a small molecule ligand, even in the absence of an *a priori* binding cavity, it will also be obvious that it must necessarily bind somewhere on the protein; either internally or externally. Furthermore, most known PAS domains have cofactors that bind to the hydrophobic core regions (see Taylor et al. 1999, p. 490, 1st column, 2nd full paragraph) making internal binding the likely choice. While it is somewhat more difficult to detect binding internally because it takes longer for binding to occur (e.g. to detect on-rates of the ligand), it still is not by any means impossible, which is a point acknowledged by Prof. Gardner. Furthermore,

binding affinities for a ligand, just because said ligand binding occurs internally rather than externally, does not necessarily correlate to the ligands true binding affinity to the protein (e.g. its K_m). Rather, said binding affinity will be a direct consequence of the ligand-protein interaction and how well the candidate ligand fits into the binding pocket or interacts with the protein's ligand-binding site residues.

Prof. Gardner asserts that the Examiner is mistaken by asserting that NMR, as taught by Fesik et al., is one of the most sensitive methods available to probe protein-ligand interactions. Rather, it is asserted that NMR is one of the least sensitive methods compared to other methods such as thermal shift assays or a PerkinElmer – AlphaScreen. Furthermore, it also requires magnitudes more protein requiring isotopically labeled protein which is economically cost-prohibitive (see point 7, pp. 2-3).

However, it is noted that Prof. Gardner subsequently does point out that NMR is a very good technique to detect low binding affinity ligands and it is one of the few methods which can give any information regarding where the ligand binding occurs on a given protein (see p. 3, 2nd full paragraph). It is asserted that these reasons rather support why one skilled in the art might be motivated to use said technique. Furthermore, the fact that it might be costly to produce PAS domains for study with NMR does not make it non-obvious to use said technique. It is noted that the fact that while a combination might not be made by businessmen for economic reasons does not mean that a person of ordinary skill in the art would not make the combination because of some technological incompatibility. In re Farrenkopf , 713 F.2d 714, 219 USPQ 1 (Fed. Cir. 1983) (Prior art reference taught that addition of inhibitors to

radioimmunoassay is the most convenient, but costliest solution to stability problem. The court held that the additional expense associated with the addition of inhibitors would not discourage one of ordinary skill in the art from seeking the convenience expected therefrom.). (see MPEP 2145).

Prof. Gardner also asserts while the Examiner discusses the effect of dynamics on NMR spectra of proteins that said Examiner is right that these effects can lead to the disappearance of peaks in spectrum of a target, but these would discourage a practitioner in the field from proceeding with further studies of a protein target. The lack of peaks in this way is highly correlated with difficulty in further analysis and screens. One skilled in the art would not proceed with screening a sample using a certain method when that method does not provide the data needed to establish if ligands are binding or not. (see point 8, bottom of p. 3).

However, the presence and absence of peaks would not necessarily discourage one of skilled in the art from using NMR. As stated, because it is known that the protein does in fact have an activity in solution, e.g. it is known that the protein does have a binding activity in solution, one need not dwell on the fact the initial NMR spectrum when the ligand is not present does not provide a ligand binding peak. Rather, it is the "after" spectrum, e.g. after the ligand has bound which will give the greatest information relative to the before spectrum because there will have to a conformational change in the protein in order to accommodate binding of the ligand and this will be revealed in the "after" NMR spectrum.

Finally, Prof. Gardner asserts that the Examiner has contradicted himself by stating that: "The major advantage of the NMR method over any other screening method is that it observes the binding of the small molecule directly to the target protein in its native environment, i.e. in aqueous solution. There is no reason to believe that the most abundant conformation in solution which is observed by NMR is the most relevant conformation for binding a small molecule or a large target molecule." Action, p. 3". It is asserted that these sentences are inconsistent wherein the first argues that NMR methods should be used for ligand screening in order to work in a protein's "native environment"; the second argues that there is no reason to expect that the "most abundant" form of the protein under these conditions should be competent to bind ligand.

However, these sentences are not necessarily contradicting. The first merely points out the fact that a huge advantage of NMR over other techniques such as protein crystallography is that NMR is done in solution rather which is the proteins native and relevant environment rather than finding a snap-shot of one of potentially many different conformational states which is given by protein crystallography. The latter statement is merely meant to draw on the fact that the most biologically relevant conformation of a protein may not be present all of the time, especially if that conformation requires co-factor or ligand binding which is required in order to induce a conformational change to said protein which results in the biologically relevant conformation.

Thus, it is asserted that the declaration and arguments presented by Prof. Gardner are unpersuasive to overcome the rejection of record.

It is also asserted that a case of *prima facie* obviousness has been established for claims 3 and 5 as being unpatentable over Fesik (WO 97/18471) in view of any one of U. S. patents 5,843,683 (Edery *et al.*); 6,291,429 (Takahaski *et al.*); or 6,436,654 (Berkenstam *et al.*) for the reasons presented herein and for the reasons already established by the BPAI on 19 September 2007.

(11) Related Proceeding(s) Appendix

Copies of the court or Board decision(s) identified in the Related Appeals and Interferences section of this examiner's answer are provided herein.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

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